# Medical Memoranda

## Carbon Tetrachloride Poisoning as a Hazard of Wig Cleaning

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One death (Veley, 1909) and seven cases of narcosis (Colman, 1907 ; Sandilands, 1909 ; Møller, 1933) have been reported after the use of dry hair shampoos containing carbon tetrachloride. Poisoning due to the inhalation of carbon tetrachloride fumes while cleaning a wig has not previously been described.

### Case Report

A 32-year-old nurse was advised by a chemist's assistant to clean her 14-in. (36-cm.) human hairpiece with carbon tetrachloride solvent. She used about two dozen cotton-wool balls soaked in the solvent, discarding them into a waste-basket. She worked in a small bedroom with a low ceiling, and heated by an oil-fired radiator. There was no fireplace, the door was closed, and, though one window was open slightly, ventilation was restricted by full-length curtains. Her health had been good, with no history of hepatic or renal disease, but 24 hours previously she had consumed several beers and some spirits at a party.

About five hours after initial exposure to the solvent she developed sudden severe upper abdominal pain with intense nausea and vomiting followed by a constricting sensation in the chest, paraesthesia in the limbs, and tinnitus. These symptoms recurred intermittently at about four-hourly intervals thereafter. Twelve hours after exposure she developed persistent low back and loin pain, with scanty foul-smelling urine.

On admission to hospital 36 hours later she had a temperature of 100.6° F. (38.1° C.) and there was tenderness in the epigastrium and right loin ; otherwise clinical examination was negative. Albuminuria and bilirubinuria were present but urine microscopy was normal. Haemoglobin, white cell count, and erythrocyte sedimentation rate were normal. Blood urea (78 mg./100 ml.) and plasma creatinine (3.3 mg./100 ml.) were raised. Blood electrolytes were normal. Carbon tetrachloride poisoning was diagnosed, with evidence of hepatic and renal involvement.

After admission she continued to have attacks of severe colicky upper abdominal pain and retching with dyspnoea and paraesthesia. Carpopedal spasm occurred on two occasions, being rapidly relieved by 10 ml. of calcium gluconate (10%) intravenously. Mild low back and bilateral loin pain persisted between the acute episodes. Oral fluids were withheld and 10% dextrose and 5% dextrose saline alternately were slowly infused intravenously.

After 24 hours the acute symptoms settled, but she remained nauseated with mild pain and tenderness in the right hypochondrium for a further two days and had marked anorexia for one week. Icterus developed on the day after admission with a high serum bilirubin (direct van den Bergh positive ; indirect 4.8 mg./100 ml.) and raised serum alanine aminotransferase at 216 units. The serum bilirubin rose to 5 mg./100 ml. 48 hours after admission, subsequently falling gradually to normal. The serum alanine aminotransferase rose to over 500 units, the serum aspartate aminotransferase to 210 units, and the zinc turbidity to 10 units after nine days, thereafter slowly falling towards normal. The plasma alkaline phosphatase remained normal throughout.

In the first 24 hours after admission 600 ml. of urine was passed, but the urinary output increased thereafter to a diuresis of over 5 litres on the tenth day, with a gradual lessening of albuminuria and haematuria. The blood urea rose to 205 mg./100 ml. on the fifth day, returning gradually to normal 22 days after admission. Associated with this was a rise in plasma creatinine to 10 mg./100 ml. The serum calcium was low on one occasion at 8.5 mg./100 ml., but several other recordings were normal.

Her fluid intake was strictly controlled until the diuresis was established, and once oral feeding was started on the third day a high carbohydrate and restricted protein diet was given. She was discharged 23 days after admission with no symptoms. The blood urea, plasma creatinine, and urine concentration test were normal, no albuminuria or haematuria was present, and the only abnormality of liver function was a slight rise in serum alanine aminotransferase (65 units). This had returned to normal two weeks later, and three months after discharge she remained symptom-free with normal urine, blood urea, and liver function tests.

### Comment

The case described presents many of the reported features of carbon tetrachloride poisoning (Dudley, 1935 ; Eddy, 1945 ; Browning, 1952 ; Guild *et al.*, 1958 ; Dawborn *et al.*, 1961), the predominant complications being transient hepatic and renal failure. Inadequate ventilation during the use of the solvent was an important factor, and recent exposure to alcohol may have resulted in impaired detoxication of the poison by the liver cells. This explanation of the increased sensitivity after alcohol, previously noted by Hammes (1941) and Guild *et al.* (1958), seems more likely than that of Lamson *et al.* (1924), who attributed it to increased absorption of the solvent.

Induced alkalosis from hyperventilation may have contributed to the paraesthesia and carpopedal spasms, but would not explain the hypocalcaemia. Susceptibility in subjects with low dietary calcium has been reported (McGuire, 1932 ; Smyth and Smyth, 1936), and in the present case intravenous calcium appeared to abort the acute abdominal symptoms.

One hairpiece cleaner has been shown to contain this solvent with lanolin, and some schools of hairdressing apparently recommend carbon tetrachloride as an alternative to proprietary cleaners, the case reported resulting indirectly from this advice. Adequate ventilation does not seem to be emphasized.

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